



Could Conservative Iron Chelation Lead to Neuroprotection in Amyotrophic Lateral Sclerosis?

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Résumé en anglais	Iron accumulation has been observed in mouse models and in both sporadic and familial forms of amyotrophic lateral sclerosis (ALS). Iron chelation could reduce iron accumulation and the related excess of oxidative stress in the motor pathways. However, classical iron chelation would induce systemic iron depletion. We assess the safety and efficacy of conservative iron chelation (i.e., chelation with low risk of iron depletion) in a murine preclinical model and pilot clinical trial. In Sod1 mice, deferiprone increased the mean life span compared with placebo. The safety was good, without anemia after 12 months of deferiprone in the 23 ALS patients enrolled in the clinical trial. The decreases in the ALS Functional Rating Scale and the body mass index were significantly smaller for the first 3 months of deferiprone treatment (30 mg/kg/day) than for the first treatment-free period. Iron levels in the cervical spinal cord, medulla oblongata, and motor cortex (according to magnetic resonance imaging), as well as cerebrospinal fluid levels of oxidative stress and neurofilament light chains were lower after deferiprone treatment. Our observation leads to the hypothesis that moderate iron chelation regimen that avoids changes in systemic iron levels may constitute a novel therapeutic modality of neuroprotection for ALS. Antioxid. Redox Signal. 29, 742-748.

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